THE ORIGIN AND SPREAD OF MYXOMATOSIS, WITH PARTICULAR REFERENCE TO GREAT BRITAIN

by Harry V. THOMPSON

Infestation Control Division, Ministry of Agriculture, Fisheries and Food, Hook Rise, Tolworth, Surbiton, Surrey.

Introduction

The first historical reference to the European rabbit *(Oryctolagus cuniculus* Linnaeus, 1758) was made by Polybius in the second century B.C., writing of Corsica. The Romans knew the rabbit and, according to Nachtsheim, they had domesticated it by the first century B.C. It was also a serious pest in the Balearic islands at this period and was well known in Spain. I have no information as to how the rabbit spread to the rest of Europe, but the first undoubted records of its presence in Britain are in the thirteenth century. The rabbit did not become a serious pest in Britain until the nineteenth century, and by that time it had been introduced into every continent and was a menace to agriculture in Australia, New Zealand and Tasmania. There are few records of the introduction of the European rabbit into the Americas, but it was released for sporting purposes on the mainland of Chile and on the Chilean half of the island of Tierra del Fuego at the beginning of the twentieth century.

The domesticated European rabbit has, of course, been used in scientific laboratories throughout the world for many years and myxomatosis was first reported in 1898 from Montevideo, Uruguay, by Sanarelli whose stock of laboratory rabbits was almost wiped out by it in 1896. Sanarelli was unable to isolate a bacterium or protozoan parasite from his rabbits and, deciding that the disease was caused by a filterable virus, he called it infectious myxomatosis. During the early part of the twentieth century there were further outbreaks of the

disease among domestic rabbits in Argentina, Brazil and Southern California (Kessel, Prouty & Meyer, 1931; Kessel, Fisk & Prouty, 1934). In many of these cases, as in virological experiments with myxoma, the mortality-rate was 99,5 per cent or higher. The clinical symptoms of classical myxomatosis in the European rabbit as produced experimentally are precisely similar to those resulting from natural infection.

There is an incubation period of 5-7 days, after which the eyes discharge a clear watery fluid. This discharge thickens in a day or two and the eyelids swell, become very thick and adhere. Swellings may appear on other parts of the body, and the bases of the ears, the nose, the feet, under the chin and the ano-genital region are frequently affected. In all cases the swellings consist of jelly-like material of connective tissue origin. Death occurs 11-18 days after infection.

The original source of the disease was a complete mystery until Aragâo, in 1942, produced evidence to show that many of the native wild rabbits of Brazil *(Sylvilagus brasiliensis* Brisson, 1766) were immune to the disease, not because of innate insusceptibility, but because they had acquired immunity from a previous infection. Those Brazilian wild rabbits that were not immune reacted to intradermal inoculation of the virus by developing a mild form of myxomatosis with no mortality.

Fenner (1952) suggested that, just as *Sylvilagus* is the New World equivalent of *Oryctolagus,* the myxoma virus is the New World analogue of the pox viruses. This hypothesis gains support from the similar pathogenesis of myxomatosis and the pox diseases, and the similarity between the myxoma and vaccinia viruses as shown by the electron microscope (Farrant & Fenner, 1953). The disease most closely related to myxoma is the fibroma of Shope (1932a), which is found in cottontail rabbits *(Sylvilagus floridanus* Allen, 1890) in the eastern United States of America. This disease causes the development of a single large tumour, as in myxomatosis of Brazilian wild rabbits, and is transmissible to domestic rabbits by implantation or inoculation. But the fibroma virus is benign in domestic rabbits and Shope (1932b) found that inoculation with fibroma acts as a form of vaccination against the lethal effects of myxomatosis.

Transmission

Myxomatosis can be spread by *direct contact* between rabbits, and will pass from an infected to a healthy rabbit kept in the same cage, but a rabbit kept in a separate cage six inches distant does not usually become infected. Droplet infection is evidently unimportant and contact infection probably takes place through a break in the skin of the healthy rabbit.

In 1920, Aragâo discovered that the cat flea *(Ctenocephalides felis* Bouché, 1835) could transmit the disease; in 1936 Torres showed that *Culex fatigans* (Wiedemann, 1828) could be a vector and, in 1942, Aragâo incriminated two other mosquitoes *(Aedes scapularis* Rondani, 1848 and *Aedes aegypti* Linnaeus, 1758). It was later found that the Australian stickfast flea *(Echidnophaga myrmecobii* Rothschild, 1909) and two Australian mosquitoes *(Aedes alboannulatus* Macquart, 1849 and *Aedes camptorhynchus)* were vectors (Bull and Mules, 1944); Mykytowycz (in Ratcliffe, Myers, Fennessy and Calaby, 1952) showed by laboratory experiments that the rabbit louse *(Haemodipsus ventricosus* Denny, 1842) and a mite *(Cheletiella parasitovorax* Mégnin, 1878) could be carriers, while transmission in the field by two other Australian mosquitoes *(Anopheles annulipes* Meigen, 1830 and *Culex annulirostris* Theobald, 1905), was shown to be very important (Ratcliffe *et al.* 1952; Myers, 1954). Laboratory studies by Fenner, Day and Woodroofe (1952), using *Aedes aegypti,* confirmed the findings of Aragâo (1943) that transmission was mechanical, since infected blood appeared to play no part. Mosquitoes were found to retain infection on their probosces for up to 25 days, while pins which had been stuck into a myxoma lesion could remain infected for 12 days. The common European rabbit flea *(Spilopsyllus cuniculi* Dale, 1878) has proved to be an efficient vector (Lockley, 1954; Muirhead Thomson, 1954; Shanks *et al.,* 1955) and laboratory studies have shown that the sheep tick *(Ixodes reduvius* L. 1758) can be a vector (Allan and Shanks, 1955). In France, laboratory experiments by Jacotot, Toumanoff, Vallée and Virat (1954) have shown that two more mosquitoes *(Anopheles maculipennis atroparvus* Van Thiel, 1927 and *Anopheles stephensi* Liston, 1901) can transmit myxomatosis. Studies in the field and the laboratory in England by Muirhead Thomson (1956 a-b) confirmed that *Anopheles maculipennis atroparvus* can transmit myxomatosis, but indicate that neither it nor the woodland species *(Aedes cantans* Meigen,

1818 and *Aedes annulipes* Meigen, 1830) are of importance as vectors in this country.

As a result of all this work it is now recognised that rabbit-biting arthropods are the principal natural vectors of myxomatosis; that any such arthropod which feeds on more than one host is a potential vector; and that, in any particular area, local and seasonal conditions will determine which vectors will be of prime importance.

Specificity

To be effective, an agent of biological control must be specific to the pest population and spread rapidly through it. Many attemps have been made to induce myxomatosis in a wide variety of animals, but all have failed. Sanarelli (1898), Rivers (1926-27), White (1929), Hyde and Gardner (1933) and others showed various species of mammals and birds — including the guinea-pig *Cavia porcellus* Linnaeus, 1758, and two American hares *(Lepus californicus* Gray, 1837, and *L. americanus* Erxleben, 1777) — to be refractory. Before field experiments in Australia were begun, Martin (1936) and Bull and Dickinson (1937) investigated a large number of species and were satisfied that all common domestic animals, rats, mice, marsupials and even the European hare *(Lepus europaeus* Pallas, 1778) were insusceptible. No other species of animals have been affected in the Australian epizootics of 1950-55, and during the European outbreaks the only animals other than rabbits to be affected have been a small number of hares (Jacotot, Vallée and Virat, 1953; Magallon, Bazin and Bazin, 1953).

In Britain, there has been much concern lest myxomatosis should spread to other animals. There have been rumours of domestic animals being affected but, on investigation, none of these has been substantiated. In two instances concerning sheep a clear alternative diagnosis was made, in one case or and in the other the condition followed the use of an anthelmintic; an alleged case of myxomatosis in a dog proved on examination to be sarcoptic mange of the head and fore-limbs (Ritchie, Hudson and Thompson, 1954). At the Veterinary Laboratories of the Ministry of Agriculture, Hudson and Mansi have examined 51 cases of suspected myxomatosis, in 16 species of mammals and birds, and with the exception of three hares none has shown any evidence of the presence of the virus. One of the affected hares was an Irish hare

(Lepus timidus hibernicus Bell, 1837) from Northern Ireland and was a clinically typical case. The other two were English hares *(Lepus europaeus)* and showed insignificant lesions.

Attempts to use myxomatosis for biological control of the rabbit

The European rabbit is a pest in many countries and, since traditional methods of control are laborious and expensive, a form of biological control has obvious attractions. One, unsuccessful, attempt was the introduction of stoats, weasels and ferrets into New Zealand in the late nineteenth century. Aragão (1927) first suggested that myxomatosis might be used to control the rabbit in Australia and sent myxoma virus to the New South Wales Department of Agriculture in 1926. Laboratory experiments (White, 1929) were carried out on the means of transmission, and the work was then transferred to Cambridge where Martin (1936) showed that the disease would spread rapidly through rabbit populations kept under semi-natural conditions in large outdoor enclosures. Extensive colony and field trials were then made in Australia from 1936-43 (Bull & Mules, 1944), and myxomatosis was found to spread effectively when rabbits were infested with the stickfast flea. Larger trials in semi-arid pastoral areas were less successful; the disease failing to spread from one warren to another, partly because foxes killed many of the infected rabbits.

About this time several attemps were also made to introduce myxomatosis into various parts of Europe. After his trials at Cambridge, Martin collaborated with Lockley in three unsuccessful attempts to establish the disease on the densely rabbit-infested island of Skokholm, Pembrokeshire (Lockley, 1940). We now know that Skokholm rabbits do not appear to have any fleas (Lockley, 1955) and the absence of this vector is the most likely reason for the failure of the experiments. Concurrently, myxomatosis was introduced on to a Danish island in the Kattegat and an estate in Sweden, but with only partial success (Jensen, 1939; Hvass and Jensen, 1939). Here again, the absence of effective vectors was probably the limiting factor.

During the war of 1939-45, the possibilities of using myxomatosis as a means of rabbit control were occasionally discussed, but dismissed as unpromising. In Aus-

tralia, however, the graziers had never lost faith in the virus and experiments conducted in 1950 by the Wildlife Survey Section of the Commonwealth Scientific and Industrial Research Organisation resulted in myxomatosis becoming epizootic in Australia (Ratcliffe *et al.*, 1952). The virus has been introduced in New Zealand and Tasmania, causing local rabbit destruction, but has not spread widely (Filmer, 1953; Meldrum, 1954).

The first successful introduction of myxoma virus into Europe occurred in June, 1952, when the inoculation of two rabbits on an estate in France led to an epizootic in 1952-53 (Delille, 1953; Jacotot and Vallée, 1953). Myxomatosis has spread from France to many European countries including Austria, Belgium, Germany, Italy, Luxembourg, the Netherlands, Spain and Switzerland (Ramon, 1953-56; Lankamp, 1953; Hase, 1954; van Koersveld, 1954) and is also the probable source of the British infection.

Myxomatosis in Britain

It has frequently been suggested that myxomatosis should be introduced into Britain, but no official attempt has ever been made to introduce the disease on to the mainland. There were a number of reasons for this decision : conditions here are very different from those in Australia; it was not known whether suitable vectors occurred here; it was doubtful if the disease would provide a permanent form of control; there was the possibility that domestic rabbit stocks would be endangered and there was a very strong bias against using a disease as a method of controlling a mammalian pest.

When it was known that myxomatosis had been successfully introduced into France and was spreading across Europe, it became evident that suitable vectors were present in the area and probably that the disease would eventually cross the Channel. The first outbreak in this country was confirmed on 13 October 1953, at Bough Beech near Edenbridge, Kent. From reports of sick rabbits seen in mid-September, it seems likely that the disease reached the district in August or early September, but the means of introduction has not been discovered; although carriage by flying or wind-blown insects, by birds carrying fleas, or by man are all possible.

In the hope of wiping out, or at least of confining the disease, it was decided to surround the affected area

by rabbit-proof netting and to kill all the rabbits within it. By 18 October the 200 acres known to contain diseased rabbits were fenced in, and by the end of the month the area was thought to be almost completely rabbit-free. Meanwhile, a second outbreak had occurred, on 27 October, near Robertsbridge in East Sussex, and the centre of this infection was wired in and the rabbits destroyed.

With the coming of winter it seemed possible that the disease might die out, but there was another outbreak on 2 November at Alciston (East Sussex), followed by others at Sevenoaks (Kent) and St. Osyth (Essex) in November, and Southwold (East Suffolk), Holland-on-See (Essex) and Lydd (Kent) in December.

It was impracticable to destroy the affected rabbits in all these outbreaks, and this procedure appeared to have little effect on the spread of the disease; except in the first four centres of infection, therefore, it was allowed to take its natural course. Myxomatosis moved very slowly during the winter of 1953-54 and by the end of February there were only eleven quite small areas of infection — all in the south-eastern counties of Kent, East Sussex, Essex and East Suffolk. At the time, this slow movement was attributed to the inactivity of woodland mosquitoes during the winter months but, as we shall see later, the spread of myxomatosis in Britain has not been a repetition of that in Australia, and mosquitoes have been of little importance as vectors.

On 3 November, 1953, three weeks after the first notification of the disease, an Advisory Committee on Myxomatosis was appointed by the Minister of Agriculture, and the Secretary of State for Scotland. The Committee presented their first report in March, 1954. They considered the history and behaviour of myxomatosis, the cost of the wild rabbit to agriculture and forestry, the trade in fur felt and rabbit meat, the claims of domestic rabbit keepers, and the effect of the disease on the rabbit. They concluded that attempts to prevent the spread of the disease from its established centres would serve no practical purpose, but that no attempt should be made to assist its spread or to introduce it into unaffected areas of the country.

There was no epizootic during the spring, and except for an outbreak in the Isle of Wight in March, myxomatosis was confined to the south eastern counties until May, when it was notified from Bedfordshire, Cornwall, Gloucestershire, Norfolk, Radnor, West Suffolk and West Sussex. Locally, the disease spread slowly, but it conti-

nued to appear in different parts of the country and by the end of June there were 78 outbreaks in nineteen counties. To be recorded as a fresh outbreak a notification of the disease had to be at least five miles from an existing outbreak. In July there were 89 additional outbreaks, which included the first two in Scotland (in the counties of Kincardine and Sutherland), 19 in Wales (six counties with outbreaks for the first time) and 68 in England (fifteen fresh counties). In August there were 88 additional outbreaks, bringing the total to 255 outbreaks in sixty-one counties, including 15 outbreaks in ten counties in Scotland. By 31 December, 1954, there were 498 outbreaks and the disease was seeded throughout the country, although large areas, particularly in the north of England and in Scotland, were unaffected. Soon after its appointment, the Myxomatosis Advisory Committee set up a Scientific Sub-Committee with a small research group, the results of whose early work was summarised by Ritchie, Hudson and Thompson towards the end of 1954.

The reports from France on the means of spread of myxomatosis (by infected herbage and motor car tyres) appeared to be at variance with the experiments of Martin (1936), which had shown that close contact was necessary for the spread of infection. Martin's work was repeated by Mr. J.D. Paterson, the Ministry's Veterinary Investigation Officer at Wye, Kent, using a strain of virus isolated from a natural outbreak. Paterson's results confirmed those of Martin. Myxomatosis passed from an infected to a healthy rabbit in the same cage, but not to another cage six inches away.

Infection was found to persist in a cage for up to twelve hours after the removal of recently dead myxomatous rabbit, but healthy rabbits did not contract the disease when fed on grass where diseased rabbits had recently been feeding, nor did they usually contract it when given food contaminated with virus. Experiments did not confirm the French belief that virus can be transported on rubber tyres.

During the early part of 1954, when the slow movement of myxomatosis was thought to be a result of the seasonal absence of adult woodland mosquitoes (then in the egg or larval stages), attention was given to other potential vectors. Since the Australian stickfast flea had been shown to be a vector, it was likely that the more mobile and very common European rabbit flea (which is absent from Australia and New Zealand) would be an effective carrier. Studies by several workers (Lock-

ley, 1954; Muirhead Thomson 1954; Shanks *et al.*, 1955) showed that the rabbit flea was an efficient vector and could remain alive on the bodies of dead myxomatous rabbits for six or seven days and still be infective. Myxomatous rabbits are known to have been eaten by foxes, carrion crows, magpies, gulls and buzzards, and predators and scavengers may have played a considerable part in the local spread of the disease. A feature of the spread of myxomatosis in Britain, however, was the large number of discontinuous jumps made by the infection. There is no doubt that the deliberate transport of infected rabbits (and their fleas) from known myxomatosis areas to other parts of the country was an important factor in the rapid dispersal of the disease which, by September 1954, was present in places as far apart as Cornwall and Shetland. The deliberate spreading of myxomatosis gave rise to much public concern, which led to an amendment to the Pests Bill (the Pests Act of 25 November, 1954) making it an offence to use a rabbit infected with myxomatosis to spread the disease among uninfected rabbits.

Another legislative measure was the making of the Non-Indigenous Rabbits (Prohibition of Importation and Keeping) Order, 1954. This arose from the fear that non-European rabbits, immune to myxomatosis, might be misguidedly introduced into Britain, to fill the niche left vacant by the European rabbit. The Order prohibits the importation into Great Britain and the keeping within Great Britain of all species of non-indigenous rabbits.

From Australian experience of four annual epizootics, in which mosquitoes were the main vectors, it was expected that in the late spring or early summer of 1954 mosquitoes and other flying insects would play a large part in the spread of myxomatosis in Britain. Althought cold weather delayed the emergence of adults in 1954, the woods were full of mosquitoes by June, but they showed little interest in rabbits. This was confirmed experimentally. Six tame rabbits were exposed to natural infection near the first outbreak of myxomatosis in Kent. Each rabbit was kept in a hutch, attached to a tree at about 5 ft. from the ground. One side of the hutch was covered only by 1 inch mesh wire netting, to allow insects to enter freely. The hutches were placed in areas where wild rabbits were dying from myxomatosis and were moved periodically to other areas of active infection. All six domestic rabbits remained in good health throughout the year.

Muirhead Thomson (1956 a) carried out studies of mosquito-rabbit ecology in the Edenbridge area of Kent and found that, although there were large numbers of *Aedes cantans* and *Aedes annulipes* present they were (with one exception) not attracted to healthy domestic rabbits exposed in the woods. He concluded that *Aedes* can have played little part in the spreard of myxomatosis in 1954, and confirmed these observations in the much hotter summer of 1955.

Soon after the netting-in and destruction of the rabbits at Bough Beech, near Edenbridge, myxomatosis appeared on a farm close by the fenced area, and then remained quiescent until January, 1954. The subsequent course of the disease in this area has been closely studied (Armour and Thompson, 1955) and plotted each week on Ordnance Survey maps. By November, 1954, the outbreak covered 375 sq. miles and had reached the limits of its possible expansion. It was bounded to the north by Greater London, and had joined up with independent outbreaks to the south, east and west. There were reports that diseased rabbits were being taken from the Bough Beech area and released elsewhere, but we believe that this had little cr no effect on the spread of infection in this area. In the places nearby, where it is possible that such rabbits were released, infection occurred only in small pockets from a quarter to one square mile in extent, and these areas were later merged in the general spread of infection.

Since we believe the spread to have been almost entirely natural in this area, it is particularly interesting to note the speed at which is moved. This varied from one to nine miles a month and averaged $3\frac{1}{2}$ miles a month from February to November, 1954. Similar rates of natural spread were observed in other parts of the country and the disease did, indeed, often travel very much slower than this. On most farms in the Edenbridge area myxomatosis took about six weeks to pass through the rabbit warrens and, although the spread was erratic, very few rabbits escaped infection. During 1955, this small residue has bred successfully and this emphasizes the need for vigorous control measures after myxomatosis. In the Edenbridge area, as in other parts of the country, the disease has lingered on almost unnoticed and sporadic cases of infection still occur.

After myxomatosis had survived its first winter and had begun to spread in Britain, it was expected that there would be outbreaks of disease among domestic rabbits, as had happened in France in 1953. The Mi-

nistry's laboratories at Weybridge began work on the preparation of Shope's fibroma vaccine within a few days of the notification of myxomatosis in Britain, and were able to arrange for commercial supplies of the vaccine to be available in April, 1954, before the anticipated summer spread of myxomatosis by mosquitoes. Although many rabbit breeders vaccinated their rabbits and screened their rabbit houses against mosquitoes, a large number took no precautions. Even so, surprisingly few outbreaks of myxomatosis among domestic rabbits were reported either in 1954 or 1955. This confirmed the view that mosquitoes were of little importance as vectors in Britain. There were, however, some cases of myxoma infection among domestic rabbits, and in several instances these were associated with the presence of the coastal mosquito, *Anopheles maculipennis atroparvus*. Muirhead Thomson (1956 b) found this mosquito carrying infection in the same hutches as myxomatous rabbits. The mosquito can live over the winter, feeding at intervals, and the virus can survive in it for over six months, but since the virus has not been recovered from mosquitoes caught outside the hutches these insects are unlikely to be important carriers.

Early in January 1955, the Advisory Committee on Myxomatosis presented its second report. They considered the spread of myxomatosis, the effect of the disease on the rabbit, the vaccination of domestic rabbits, and other matters, and recommended that every advantage should be taken of outbreaks of myxomatosis to eliminate rabbit survivors and prevent them from building up in numbers again. Throughout 1955 myxomatosis continued to spread, maintaining its high virulence and causing a mortality of about 99 per cent — except in two places mentioned later. By the end of the year, the disease was present throughout England, Wales and Scotland, although in some places where rabbits had never been numerous there were quite large areas to which it had not penetrated.

Some attenuation of the myxoma virus in Europe had long been expected and was first noted in April, 1955, in England (Hudson and Mansi, 1955; Hudson, Thompson and Mansi, 1955) and also in France (Jacotot, Vallée and Virat, 1955). Strains of virus were sent from both countries to Fenner in Australia who has compared them with Australian strains (Fenner and Marshall, 1955). In Australia, strains of high virulence are now rarely recovered in the field and a virulence giving a 90 per cent mortality is normal; the French

and English strains of reduced virulence are at least as attenuated as the Australian field strains.

Ever since myxomatosis was first notified in Britain, in October 1953, rabbits surviving outbreaks of the disease have been examined for evidence of recovery. Most of these rabbits have been found to be susceptible, and must have escaped infection in the wild; but a number of them had recovered from myxomatosis and had immune bodies in their serum. From some apparently recovering rabbits, strains of virus of normal virulence have been isolated. But in parts of Sherwood Forest, Nottinghampshire, from April 1955 onwards, estate workers and the Ministry's field officers noticed that an unusual number of rabbits appeared to be surviving myxomatosis. The disease was first notified in Sherwood Forest in September 1954, and on some estates the mortality was low despite the presence of numerous rabbits and rabbit fleas. Strains of virus isolated from recovering rabbits caught in this area produce a « nodular » type of infection, when inoculated into domestic rabbits, and infected wild rabbits bear similar nodules. These small nodules may cover extensive areas and appear anywhere on the body, although mostly on the face and ears. It is curious that during the early stages, the nodules contain typical fully-virulent virus, but later when the nodules shrink and form scabs the virus is of reduced virulence. Attenuated virus has been recovered from one other place in Britain, near Winchester in Hampshire. Unlike the Sherwood Forest area, most of the rabbits near Winchester were killed by normally virulent myxomatosis, and it is only a part of the residual population which is affected by a nodular strain of the disease. Both in Sherwood Forest and in Hampshire the rabbits surviving the attenuated strains of myxomatosis are being dealt with by rabbit clearance schemes, in which farmers, landowners and Government bodies are co-operating. An interesting suggestion made by Hudson (1956) is that these attenuated variants may have been arising all the time, and that the occasional immune rabbit recovered in the original outbreaks survived because it had been infected with a less virulent virus, rather than because of its innate resistance.

After the fine summer of 1955 there were excellent harvests of many crops, at least partly owing to the absence of rabbit damage. Very heavy crops of hay were cut, cereal crops grew right up to the hedges and many farmers were able to enlarge their beef or dairy herds. While it is difficult to evaluate the different fac-

tors, such as weather and the absence of rabbits, statistically, the net gain from cereals alone is estimated to be about £15 million and the yield from pastures and leys may well exceed this figure.

It is now almost impossible to find a really heavy rabbit infestation on the mainland of Britain, although some moderate ones exist and occasional rabbits, or pockets of rabbits, are found in many places. Myxomatosis has spread to nearly all the formerly rabbit-infested parts of the country, but it must be remembered that rabbits which survive an attack by a weakened strain of the virus are immune to the fully virulent virus and unless standard measures of rabbit control continue to be applied, the present freedom from rabbits will not be maintained. The Pests Act, 1954, authorised the designation of Rabbit Clearance Areas and the greater part of the country is now covered by such Clearance Areas. The scheme has met with a keen response from the agricultural and forestry communities and it is now usual for entire counties to be designated as such areas, which are to be kept, so far as is practicable, entirely free from wild rabbits. Although the responsibility for their destruction falls upon the occupier, the Ministry and the County Agricultural Executive Committees are helping in many ways, including the provision of grants in aid of scrub-clearance, the bulldozing of warrens, rabbit fencing and the destruction of rabbits on common land. The complete elimination of the rabbit would be an undoubted gain to our economy, and it is only by aiming at extermination that its numbers will be kept within bounds.

The spread of myxomatosis has raised many interesting problems in virology and there is still a great deal to be found out about the mechanism of spread in this country. Very little is known about the life history of the rabbit flea and some research is urgently needed. The indirect effects of myxomatosis, and the reduction in rabbit numbers, upon other animals and upon plants have been considerable and are receiving much attention, but a consideration of them is outside the scope of this paper.

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